A Rare Case of Multiregional Spinal Stenosis: Clinical Description, Surgical Complication, and Management Concept Review

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Abstract

Study Design Case report and literature review.

Objective Multiregional spinal stenosis (MRSS) has not been described in the English literature, although a few studies report the concept of tandem spinal stenosis. Due to the concurrent spinal stenosis occurring in three separate regions of the spine, clinical presentation of MRSS may be less distinct, and its surgical treatment priorities and challenges differ from single-region spinal stenosis. The purpose of this article is to describe a new concept and a rare case of MRSS as separated segments of spinal stenosis in the cervical, thoracic, and lumbar spine.

Methods A retrospective case description of MRSS and surgical strategies used in managing such extensive multiregional stenosis and its potential complications.

Results A novel surgical strategy using a combination of laminectomies with fusion and laminoplasty without fusion to treat this patient with such extensive cervical to thoracic myelopathic cord compression is described. Initial good recovery after cervical cord decompression was followed by a delayed recurrence of symptoms from thoracic cord compression. The subsequent thoracic surgical decompression, its complications and management, and patient recovery are discussed with a literature review highlighting the possible mechanisms for postoperative loss of neurologic function after thoracic decompression.

Conclusion MRSS is a rare cause of extensive compression of multiple regions of the spinal cord. To the best of the authors’ knowledge, this report is the first to use the term multiregional spinal stenosis to describe this new emergent clinical entity, surgical management strategies, and potential complications.

Introduction

Multiregional spinal stenosis (MRSS) is defined by the senior author as spinal stenosis occurring concurrently in all three regions of the spine including the cervical, thoracic, and lumbar spinal segments. This condition can be confusing to clinicians as it can present with patchy distribution of upper and lower limb motor and sensory symptoms. In this case report, we aim to discuss a case of MRSS from degenerative changes, ossified posterior longitudinal ligament (OPLL), and ossification of the yellow ligament (OYL). In addition, this report aims to describe the clinical presentation, surgical treatment, and complications of MRSS.

Case Report

A 36-year-old man with a past medical history of type 2 diabetes mellitus, hypertension, asthma, and epilepsy...
presented to us with a 7-month history of atraumatic thoracolumbar back pain with right lower limb radicular pain, numbness, and weakness. His symptoms deteriorated over the previous 3 months, and he complained of unsteady gait with frequent falls. On the Medical Research Council (MRC) scale for muscle strength, his right hip flexion and knee extension were grade 3 and 4, respectively. He was hyperreflexic in the lower limbs. Lhermitte sign was positive with electrical sensation running down all four limbs. This was associated with a positive Romberg test, and inability to perform tandem gait. There was no bladder and bowel dysfunction.

Whole-spine magnetic resonance imaging (MRI) and computed tomography showed multiregional spinal stenosis with extensive mixed type OPLL from C2 to T2 and an OYL at the level of T1/2. There were T2-weighted MRI cord signal changes at the T1/2 spinal level. OPLL was also found at the T4–6, T8/9, T10–L2 levels causing moderate spinal stenosis.

In addition, degenerated intervertebral disk bulging at the L4–S1 level was noted, resulting in mild spinal canal stenosis (►Figs. 1 and 2). Electromyography (EMG) did not show any denervation changes, and nerve conduction study (NCS) showed normal conduction for all nerves except mild right-sided carpal tunnel syndrome.

The patient underwent surgical treatment with a novel strategy combining C1–3 laminectomy, posterior instrumentation and fusion, C4–6 open-door laminoplasty, and C7–T2 wide laminectomy with posterior spinal fusion and instrumentation with OYL resection. Postoperatively, his recovery was unremarkable except for transient right C5 palsy. Lhermitte sign and right lower limb weakness were resolved. There was mild residual right lower numbness. He was discharged ambulating after short-term inpatient physiotherapy.

This patient was well until 10 months later when he was readmitted to the hospital after an atraumatic acute...
exacerbation of back pain with unsteady gait, right lower limb weakness, radicular pain to the right anterior thigh, and patchy numbness of the bilateral lower limb, which started 1 month prior to his readmission.

On examination, his upper limb function was normal but he had reduced power on right hip flexion and knee extension (MRC grade 3), dorsiflexion, hallux extension and flexion (MRC grade 2), hyperreflexia of ankle and knee reflex, and decreased sensation from the level of T10 downward. He had positive Rhomberg test and was unable to perform tandem gait.

Repeat whole-spine MRI showed a well-decompressed cervical and upper thoracic spine (Fig. 3). There was mild spinal canal stenosis with OPLL abutting the spinal cord at T4–6 levels, a large central disk prolapse, and OPLL at T8/9 with cord compression at T10–L2 levels and a relatively unchanged degenerative disk disease with mild L4–S1 stenosis noted. EMG and NCS did not show any change from previous findings.

The patient underwent elective lower T7 to upper T9 laminectomy, lower T11 to upper L2 laminectomy, and posterior spinal instrumentation and fusion from T8 to L2 with good initial recovery. The spinal drain was removed with less than 50 mL output in the drain on postoperative day (POD) 4. He was transferred to a rehabilitation hospital on POD 5. He was readmitted with a sudden deterioration of lower limb power and became paraplegic (grade 0) with urinary retention on POD 12. Urgent repeat MRI showed epidural hematoma compressing the cord from T8–9 and T11–L2. The patient underwent emergency wound exploration and hematoma drainage in the same day but postoperatively, he remained paraplegic. In view of the poor recovery (grade 0), the patient underwent further decompression laminectomies from T6 to upper L3, epidural adhesiolysis, and posterior spinal instrumentation and fusion from T6 to L3 to enhance spinal construct stability and reduction of thoracic kyphosis aimed to allow better indirect cord decompression through dorsal spinal cord migration after the laminectomies (Fig. 4). Post–revision surgery, this patient remained paraplegic (grade 0) for 4 to 6 weeks and then lower limb motor power gradually returned. His right lower limb power was grade 4 and left hip flexion grade 3, knee extension grade 4, and dorsiflexion, hallux extension, and flexion were grade 2 at 3 months after the last surgery, and the patient was discharged home.

At 3.5 years after the cervical spine surgery and 2.5 years after the thoracic surgery, he is able to ambulate with a walking stick. He has occasional neck and backaches but...
there are no more radicular symptoms or numbness in all four limbs. Bladder and bowel functions are normal.

**Discussion**

MRSS is defined as separated regions of spinal stenosis occurring concurrently in all three segments of the spine including cervical, thoracic, and lumbar spine. MRSS has not been described although a few studies reported concurrent cervical and lumbar spinal stenosis. Teng and Papatheodorou in 1964 were first to describe an entity known as concurrent cervical and lumbar stenosis, and it was subsequently termed as tandem spinal stenosis (TSS) by Dagi et al in 1987. MRSS may be considered as a separate entity from TSS as it appears to result from a combination of degenerative changes and OPLL and OYL pathologies. Patients with continuous spinal canal stenosis in the entire spine resulting from congenital stenosis or stenosis from achondroplasia were excluded as MRSS.

In this patient with MRSS, our surgical strategies aimed to (1) identify the single spinal region with the most severe canal stenosis and (2) match the patient's signs and symptoms to the region of spinal canal stenosis. Because the patient showed positive Lhermitte sign, myelopathic gait, and MRI evidence of severe stenosis in the cervicothoracic spinal region, the senior author decided to decompress the cervicothoracic spine as the first priority. In addition, this was to avoid sudden paralysis resulting from endotracheal intubation during thoracolumbar decompression surgery in patients with concurrent untreated cervical spinal canal stenosis.

Because our patient was young with minimal neck pain but extensive OPLL and OYL with degenerative spinal stenosis, the senior author engaged a novel surgical strategy including a combined laminectomy with fusion and laminoplasty to decompress his cord and preserve his spinal motion. The patient underwent C1–3 laminectomies with instrumentation and fusion, C4–6 open-door laminoplasty, and C7–T2 laminectomies with instrumentation and fusion with resection of OYL. Postoperatively, the patient had good recovery for 8 months.

Despite the good recovery in the beginning, our patient presented the second time with deterioration of symptoms and underwent lower T7 to upper T9 laminectomy, lower T11 to upper L2 laminectomy, and posterior spinal instrumentation and fusion from T8 to L2 with initial good results and partial return to normal lower limb power. Unfortunately, at POD 12, he had sudden loss of power in both lower limbs secondary to postoperative epidural hematoma confirmed on MRI. Despite the urgent hematoma evacuation within the day of presentation, our patient's neurologic status remained the same.

We postulate that the delayed hematoma presentation may have been a result of minor injury during patient rehabilitation. This might have been due to poor thoracic spine implant stability after multiple levels of full laminectomies. In addition, the physiologic thoracic kyphosis or even worsening of thoracic curvature after laminectomies further reduced the cord tolerance to the injury from epidural hematoma and spinal micromotion.

With these considerations, our patient underwent revision posterior decompression from T6 to L3, increased implant density by instrumentation and fusion in longer segments with wider laminectomies, and reduction of thoracic kyphosis by using hypokyphotic contoured rods and sequential intersegmental instrument compression. Postoperatively, lower limb motor power began to return from 4 to 6 weeks after surgery, and the patient was able to lift his legs at 3 months with subsequent ambulation with a walking stick at 1 year after revision surgery.

Based on literature review and our experience, we propose that if posterior decompression is to be used for thoracic myelopathy from OPLL or OYL, long multisegment with wide pedicle-to-pedicle decompression laminectomies and high-density spinal instrumentation should be considered. We also recommend reduction of kyphosis to allow more backward shift of the spinal cord for reduction of ventral compression by OPLL. In cases with multilevel thoracic decompression surgery, we suggest longer duration of low-suction drains, and detailed serial postoperative examination of the patient should be performed to identify early neurologic deficits.

Our recommendation is limited due to lack of statistical power, and further studies are required to define MRSS and validate our treatment strategies.

**Conclusion**

This case report has described a rare cause of a patient suffering from extensive compression of multiple regions of the spinal cord. To the best of the authors' knowledge, this report is the first to use the term multiregional spinal stenosis (MRSS) to describe this emergent clinical entity. In addition, this report described a successful novel surgical strategy using a combination of laminectomies with fusion and laminoplasty without fusion to treat a patient with such extensive cervical to thoracic myelopathic cord compression. The report also highlights the dangers in treating thoracic spinal cord compression and describes the possible mechanisms for postoperative loss of neurologic function, its management, and its history to its recovery.

**Disclosures**

Choon Chiet Hong, none
Ka Po Gabriel Liu, none

**References**